ABSTRACTS 403

\* ADENOSINE TRANSPORT BY LUNG. Harry Steinberg, M.D. and Dipak K.

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Adenosine triphosphate and adenosine monophosphate are hydrolyzed to adenosine on the surface of the pulmonary endothelial cell by 5' nucleotidase. Adenosine, a nucleoside and potent vasodilator, is taken up by the lung and converted by deamination to inosine. Although nucleoside transport has been examined in other cell systems, it has not been clearly defined in lung tissue. To study nucleoside transport, we used an isolated, ventilated rat lung perfused with Krebs-Ringer bicarbonate containing bovine serum albumen. In a single circulation through the lung, 60.8±2.5% of infused (14C) adenosine (1µM) was removed from the circulation whereas 22.5 $\pm$ 2.4% of ( $\frac{14}{C}$ ) adenine ( $1\mu M$ ), a purine base, was removed. In a single circulation,  $28.3\pm3.6\%$  of infused ( $^{14}C$ ) inosine ( $^{1}\mu M$ ), an analog of adenosine, and  $25.6\pm1.8\%$  of infused ( $^{14}C$ ) hypoxanthine (luM), an analog of adenine, were taken up by the lung. Dipyridamole, (10-8M) markedly reduced the pulmonary uptake of adenosine and inosine but failed to have an effect on adenine or hypoxanthine uptake. Moreover, inosine (10µM) was able to inhibit adenosine uptake by 68.5±2.9% whereas adenine or hypoxanthine had no effect on adenosine uptake. In the presence of inhibitors of adenosine deaminase, metabolism of adenosine was virtually abolished but adenosine transport out of the pulmonary circulation proceeded normally. These results suggest that a specific and rate limiting transport system may exist in the lung for adenosine as well as other nucleosides and, as in other cell systems, this transport capability may have importance for cell growth and malignant transformation. (Supported in part by NIH #R23 HL 18355)

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